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Feline acromegaly: how to recognize it? Is it frequent?

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Acromegaly is typically diagnosed in elderly cats. There appears to be a clear male predisposition, as nearly 90% of affected cats are male or male-castrated. Of note, there is also a male predisposition for diabetes in cats in general, i.e. 70-80% of diabetic cats are male. The disease is caused by a growth hormone (GH)-producing tumor (usually an adenoma) in the pars distalis of the pituitary gland. GH has catabolic (diabetogenic) and anabolic effects; the latter are in part mediated by insulin-like growth factor-1 (IGF-1). The earliest and most common clinical signs usually are polyuria, polydipsia and polyphagia associated with GH-induced diabetes mellitus. Polyphagia may also develop as a direct effect of the GH excess and can become extreme. Often, initial weight loss is followed by a period of stabilization and thereafter, slow and progressive weight gain develops. During the first few weeks or months after initiating therapy, insulin requirement may be relatively low (i.e. 1 – 3 units per cat BID) and then starts to increase as the insulin resistance worsens. Insulin requirement may easily exceed 1.5 – 2.0 U/kg body weight BID and the use of 30 units BID and more has been reported. A cat with difficult to regulate diabetes and weight gain at the same time should alert the clinician to consider the possibility of acromegaly. The physical changes of acromegaly usually have an insidious onset and progress very slowly. The soft tissue overgrowth and the osseous changes cause weight gain, enlargement (broadening) of the head, enlargement of the mandible (prognathia inferior), widening of the interdental spaces and sometimes a large tongue. Diffuse thickening of oropharyngeal tissue can lead to respiratory stridor and respiratory distress. Enlargement of the abdomen and the paws (“clubbed” paws) are also identified regularly. The general impression is that of a large cat. Degenerative arthropathy due to proliferation of chondrocytes and disruption of joint geometry is common, as is systolic heart murmur and possibly cardiomyopathy. Organomegaly also involves liver, kidney, spleen and pancreas as well as other endocrine organs such as adrenal glands, thyroid and parathyroid glands. CNS signs as a result of pituitary tumor growth are seen in approximately 10 – 15% of cats. Laboratory work-up reveals findings typically associated with diabetes mellitus. Hyperproteinemia seems to be the only parameter being more frequently present in diabetic cats with acromegaly compared to diabetic cats without acromegaly. Hyperphosphatemia (without azotemia) is occasionally seen.

Acromegaly has long been considered a rare disorder. However, recent studies show that the prevalence may have been underestimated. According to our studies in Zurich we assume that 10 – 15% of diabetic cats suffer from acromegaly. Diagnosis requires the demonstration of GH excess and/or high IGF-1 concentrations. Since the availability of a validated GH assay for cats is a problem, IGF-1 has evolved into a popular test to screen for acromegaly. Its concentration is quite stable throughout the day requiring just a single blood sample which can be sent by regular mail. It should be kept in mind that IGF-1 results may be false negative and false positive. False negative results may be seen in early stages of the disease, serious concurrent disease (e.g. lymphoma) and in some acromegalic cats if the measurement is done before the start of insulin therapy. False positive results may be found with the use of some assays which remove the IGF-1 binding proteins only incompletely (intra-assay inference of binding proteins). It is also possible that in some diabetic cats aggressive and/or long-standing insulin therapy results in false increase in IGF-1 which is not associated with acromegaly. Acromegaly should be considered in all cats with difficult to regulate diabetes, in which problems associated with the insulin and the insulin administration, short duration of insulin effect and other common concurrent problems (e.g. glucocorticoid administration, severe stomatitis/gingivitis, urinary tract infection) have been excluded. The finding of a high IGF-1 level would support the suspicion of acromegaly and should be followed by imaging of the pituitary gland by CT or MRI. A pituitary mass is visible in the vast majority of cases at the time of presentation. The finding of a normal IGF-1 concentration, on the other hand, does not rule out acromegaly. IGF-1 measurement should be repeated after a few more weeks of insulin therapy (or 6 – 8 weeks after insulin therapy has been initiated), or an immediate search for a pituitary mass by CT/MRI may be considered.

Experience with treatment is limited. The most frequently used modality is radiation therapy. We and others have seen clinical improvement, reduction of insulin requirement and decrease in size of the pituitary tumor. However, transsphenoidal hypophysectomy seems to be superior to radiation therapy as diabetic remission may be achieved in most (if not all) cases. Pituitary surgery is a highly demanding procedure and availability so far is limited.

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